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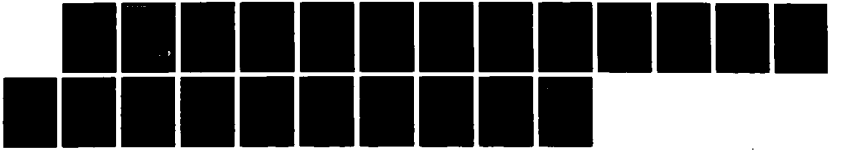

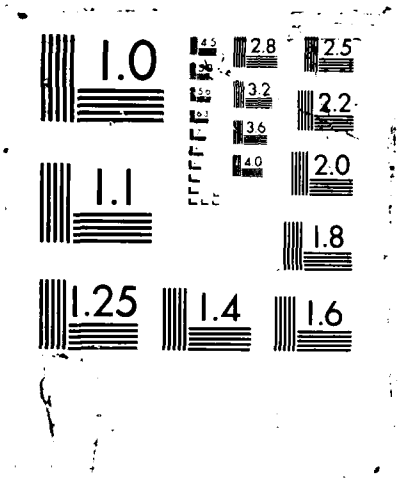
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MURRAY P. HAMLET, D.V.M.
Director, Cold Research Division

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Cold injuries have played a major role in the defeat of armies throughout history. The impact on medical treatment facilities is staggering and Command influence on prevention is essential to an Army's functioning in the cold. The non-freezing injury of immersion foot or trench-foot occurs for relatively small numbers of people in training but can be significant in a combat setting. Frostbite, the actual freezing of tissue, is the major threat to the wounded and to individuals unprepared to deal with the cold environment. The risk factor recognition, current methods of treatment and management of these injuries are critical for a military surgeon who must advise the Commander.

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WAR SURGERY HANDBOOK

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HISTORICAL ASPECTS:

Cold injuries have had a major role in the outcome of a number of military operations throughout history. Larrey's description of the loss of over 250,000 soldiers in Napolean's Army in Poland in 1812 identified cold as the major force in defeating this grand army. In the Crimean war, during 1852-1856, 309,000 French troops experienced 5,215 cases of frosbite of which 1,178 were fatal. In just two nights, in Sevastopol, 2,800 cases of frostbite occured with deaths. In WWI, the British incurred 115,000 trenchfoot or frostbite injuries. In one, six-week period there were 1,131 men with frostbite in one hospital in Rouen. In the Dardanelles, from April, 1915 to January, 1916, there were 14,584 admissions for cold injury. Although U.S. Army losses in WWI amounted to only 2,061 admissions. This resulted in a total of 97,200 man days lost. In just 2 months in WWII, November and December of 1942, the German army suffered 100,000 cold injuries requiring 15,000 amputations. This was a major factor in their defeat on the eastern front. U.S. experiences in WWII and Korea revealed that 10% of the wounded casualties (90,000 in WWII and 9,000 in Korea) were cold injuries. Recent British experience in the Falklands listed trenchfoot as the major medical problem in that conflict. Argentine amputations in the same conflict exceeded 200. The lessons on the impact of cold on military operations appear to be relearned each time an Army is subjected to cold or cold/wet situations. Any force poorly fed, poorly clothed and in retreat is more likely to sustain serious cold injuries. Adding to the Command problems of prevention of cold injury, the medical personnel providing care are often unaware of the seriousness of the threat and have little or no experience in dealing with cold injuries.

CLASSIFICATION:

Cold is the primary etiologic agent in producing these injuries, although wetness, duration of exposure and other concomitant injury may add to the severity or eventual outcome of a particular cold exposure. Long-term exposures in wetlands, even tropical areas, rice patties, swamps and jungles with long-term cooling of the feet and constant wetness can produce an immersion type injury. Cold injuries offer a continuum of insults making the definition between one type and another rather artificial. The following definitions are of both historical and clinical significance and are in general use. (1) Chilblain - frequently affects the hands and the feet and may result from exposure to air temperatures from above freezing to as high as 60°F, (16°C), and often is associated with high humidity. It is not of major clinical significance to military operations. (2) Immersion foot - applies to injury caused by exposure, usually in excess of 12 hours, to water at temperatures generally above 50°F, (10°C). This is a common life raft injury. (3) Trenchfoot - which also may occur on the hands, results from prolonged exposure to cold at temperatures ranging from just above freezing to plus 50°F (10°C), often in a damp environment and usually in connection with immobilization and dependency of the extremities. Historical implication of blunt trauma, such as walking on wet limbs, in the production of trenchfoot injuries should be noted. (4) Frostbite - implies the crystalization of tissue fluids in the skin or subcutaneous tissues after exposure to temperatures of 32°F (0°C) or lower. Depending on the ambient temperature and the wind velocity, the exposure necessary to produce frostbite varies from a few minutes to several hours.

Frostbite may occur at various altitudes. This is the most common cold injury in military operations. (5) Hypothermia - a condition associated with a drop of core temperature below 94°F (34.4°C) This life threatening, non-freezing, cold injury is usually the result of long term exposure to either cold air or immersion in cold water. It should be noted that freezing temperatures are not necessary to produce hypothermic victims as wind, rain and cool temperatures significantly increase body heat loss.

PATHOGENESIS:

Trenchfoot, frostbite, immersion foot, and hypothermia are the cold injuries of greatest military significance. It is believed that frostbite will continue to be the cold injury of major importance, but the advent of rubberized, insulated, footwear and specific training techniques and procedures will limit the trenchfoot injuries in future combat settings. It is noted that the introduction of the insulated vapor barrier boot to U.S. forces in Korea almost eliminated cold injury as a reason for hospital admission. This insulated vapor-barrier boot revolutionized footwear for combat soldiers and was a key for prevention of cold injury. The type of cold injury is dependent upon the temperature at which the exposure occurs, the duration, and environmental factors such as wind and water, which intensify the effect of the temperature. On exposure to cold, there is an initial peripheral vasoconstriction in an attempt to conserve core heat. This vasoconstrictive eposide lasts a short period of time until a physiologically protective mechanism termed CIVD (cold-induced vasodilation) intervenes to cause arteriovenous shunting to improve skin

temperature. This allows relatively large volumes of blood to flow through cold extremities. Repeated cold exposures are said to improve this CIVD response, but it may be suppressed or absent when the individual is chilled, frightened, tired, or hungry. This mechanism appears blunted in Blacks and perhaps in other races. Trenchfoot and immersion foot are essentially the same injury; the major difference being the duration of exposure and the temperatures involved. The colder it is, the shorter the duration necessary to produce trenchfoot; the longer the duration and the warmer the temperature, the more likely you are to have an immersion foot type injury. The average duration of exposure for trenchfoot is 3 days, but may range from a few hours to several days. Individuals immersed in seawater even for a period as short as 2-3 hours may have significant immersion injury. Individual susceptibility varies significantly. The average duration for frostbite production is 10 hours, but temperature, moisture, clothing, activity and other factors including individual training may vary this significantly.

PATHOLOGIC PROCESS:

Although cold is the single most significant factor involved in the destructive process of cold injury, intracellular molecular changes due to hyperosmolality as ice crystals form and direct metabolic impairment of cell function seem to play a small role in the total destructive process. In non-freezing, cold/wet injuries changes in membrane permeability of the most sensitive cell types, nerve, muscle and endothelium appear to be the most critical part of the injury process. In freezing injuries, ice

crystal formation and the destructive force on capillaries and endothelial cells lining small vessels appear to have the major role in cell death. The loss of tubular integrity of the vascular system decreases nutritive flow supporting the clinical and experimental data indicating that capillary flow appears to be the determinant factor in the irreversibility of frostbite injury. Destruction of capillary networks, plugging of small blood vessels, and complete thrombosis of larger vessels delineate the degree of frostbite injury.

HYPOTHERMIA:

Whole body cooling associated with decreasing oxygen supply and cardiovascular failure lead to complete circulatory collapse and death from hypothermia.

EPIDEMIOLOGIC FACTORS:

The military community responds to cold trauma according to accepted epidemiologic principles. The specific causative agent is cold. Moisture and wind add to the threat as both increased conductive and convective heat loss. Cold increases the rate of body heat loss in hypothermia, but can also produce ice crystals within the tissue in frostbite.

ENVIRONMENTAL FACTORS:

A variety of environmental and host causation factors influence the incidence, prevalence, type, and severity of cold injury, though this influence varies from situation to situation. The most important

environmental factors in cold injury are weather, clothing, and the type of combat action. Weather has a predominant influence in the causation of cold injury. Temperature, humidity, precipitation and wind modify the rate of heat loss from the body. Low temperature and low relative humidity favor the development of frostbite. Higher temperatures, together with moisture favor the development of trenchfoot. Wind and moisture favor the production of whole body heat loss for hypothermia. The wind velocity and low temperatures interact, as described by wind chill, to accelerate the loss of body heat under conditions of both wet and cold. The type of combat action appears to be a most important environmental factor. Units in reserve or in rest areas have few cases. Units on holding missions or in static defense in which exposure is greater show a moderate increase in incidence. Factors which modify the incidence in relation to the rate of combat include immobility under fire, prolonged exposure, lack of opportunity to warm the body, change clothing or carry out personal hygiene measures, along with mental and physical fatigue, fear, and the state of nutrition. The rate of combat, the losing side in retreat, those faced with an overwhelming foe, or an unrelenting wind during combat all add significantly to the risk of major cold injuries. In warfare in which exposure under conditions of stress may be prolonged, adequate clothing becomes essential to welfare and survival. British experience in the Falklands in which well-trained, knowledgeable soldiers suffered extensive trenchfoot injuries because the combat setting posed an inability to use preventive measures, was a clear indication of the role that the combat setting has on the production of injury.

HOST FACTORS:

The following host factors influence the development of cold weather injury. (1) Age - because of the similar age of combat troops, there is little evidence that significant differences in susceptibility exist. Age does, however, impart a level of experience in combat. This experience decreases combat stress and increases the individuals ability to deal with prevention of these injuries. (2) Smoking - there is very clear evidence that the vasoconstrictive action of nicotine causes increased cooling of the extremities and increase the likelihood of frostbite. A significant number of severe injuries seen in military populations occur in heavy tobacco users. (3) Previous cold injury - individuals with previous cold injuries are at higher than normal risk of subsequent injury. Repeated minor cold injuries often produce abnormal peripheral cold sensitivity similar to Raynaud's. However, repetitive injuries often do not occur at the same site, indicating that this may be an individual susceptibility rather than modifications in circulation or local resistance to cold from a previous injury. (4) Branch of Service - Trenchfoot and frostbite have a selectivity for frontline riflemen. This relates to the type and duration of exposure and makes them a high risk group. (5) Fatigue - Both mental and physical weariness contributed to apathy which leads to neglect of acts, except those acts vital to surivival. Fatigue is most evident in troops who are not rotated and must remain exposed and in combat for long periods of time. Three days of being cold and wet appears to be a significant timeframe for consideration of rotation of troops. (6) Racial susceptibility - In all studies concerning WWII, Korea, and recent

experience in Alaska, blacks had four to six times the incidence of cold injury as did their caucasian counterparts matched for geographic origin, training, and education. This increased succeptibility is related to two factors: (a) differences in anatomic configuration, and (b) differences in physiological response to cold. Because long, thin fingers and toes cool more rapidly than short, fat ones, blacks' hands tend to cool faster than those of caucasians. More important, however, once cold, Blacks stay colder longer because of the lack of significant CIVD response in their extremities. This does not say, however, that Blacks cannot be protected from cold injury. Commanders and Blacks themselves must be more vigilant during cold exposure and must take measures sooner to protect themselves from cold injury. Home of origin has a significant role in cold injury susceptibility. Individuals raised in northern tier states, i.e., cold climates, have a more protective CIVD response. This also improves in blacks from northern climates. This is not only a physiological improvement in response to cold, but a behavioral response as well. Knowledge of clothing use, knowing when one's extremities are too cold, not being frightened of the cold and knowing how to deal with cold extremities all add up to make cold experienced individuals less likely to have cold injuries. Individuals with labile vasomotor conditions such as Raynaud's are also susceptible to cold injury.

OTHER INJURIES:

Concomitent injuries associated with reduction in circulating volume or localized reduction in blood flow increase susceptibility to cold injury.

Transport of battle injured individuals increases their likelihood of frostbite injury unless additional insulation or auxiliary heat is provided. Dehydration plays a major role often causing hypovolemia with decreased circulation to the extremities. Of minor significance could be cold urticaria, cryoglobulinemia, cryoproteinurin and other coagulopathies.

DISCIPLINE TRAINING AND EXPERIENCE:

Cold injury is preventable. Well-trained, fit, disciplined men can be protected from cold injury even in adverse, pinned down positions if they are knowledgeable concerning the hazards of cold exposure and informed regarding the importance of personal hygiene, care of the feet, exercise and the rational use of clothing. Such discipline and training are a command and not a medical responsibility. Reinforcement of these principles throughout the field operations is essential to the goal of protection from cold injury. Although cold injury is preventable, commanders may be faced with circumstances which are likely to lead to large numbers of casualties. A decision may have to be made to take a certain number of cold injuries to win the battle under some conditions. Conducting a major offensive in a cold, wet environment during a retreat or if faced by an overwhelming foe the Commander might opt to accept cold injuries to change the tide of battle. Medical implication for such decisions must be carried to the highest levels of command. The combination of fit, disciplined soldiers with cold weather training along with Commanders who provide dry clothing, food, water, and adequate shelter help assure a minimum number of cold injuries.

DRUGS AND MEDICATIONS:

Any drugs which modify central temperature regulation, autonomic system responses, or that alter sensation or judgement can have disasterous effects on individual performance and safety in the cold. Medical personnel must be alert to the role these drugs play in increased susceptibility. Unit commanders must be informed of the high risks associated with drug or alcohol use. Alcohol use, for example, is the highest single leading factor in production of hypothermia in a civilian community. The combination of altitude (hypoxia) and cold also add significantly to the production of cold injuries.

CLINICAL MANIFESTATIONS:

The progression of clinical manifestations may begin with what patients generally describe as initial feelings of cold discomfort in their extremities followed by varying periods of pain and mild discomfort along with a cyclic, dull ache. These symptoms subside into a period of anesthesia. From there, cold injury progresses in a painless fashion. Patients often describe a sensation of walking on a wooden limb. Because of this, the anesthetic nature of cold injury, patients often say they were unaware that they were getting an injury. The hypothermia victim retreats inward and senses are dulled. The victim develops a stumbling gait, loss of muscle coordination, along with slurred speech, and is most universally unaware of this insidious onset. In a cold, wet environment, trenchfoot often appears. Anesthesia of the limb in trenchfoot injury comes on rapidly. Subsequently, pain which does not respond to analgesia, limits

deployment of soldiers although extremities appear normal. Most patients are unaware or don't care about the impending severity. The first physical manifestation of frostbite injury is reddening of the skin which later becomes pale, waxy white, and hard. Lack of mobility of skin over joints is a common finding. In hypothermia, shivering is a clear indication of loss of body temperature. Shivering varies with age, physical condition, the degree of hypothermia and the amount of injested drugs. Shivering can significantly limit individual performance of specific military tasks including sighting, map reading, and manipulation of small dials and radios. It is a form of involuntary exercise that produces heat; however, when shivering stops the patient is at the mercy of the environment. CNS involvement appears the most common outward manifestation of hypothermia. Decreased dextrity and coordination, speech and memory impairment, along with eventual loss of consciousness indicate progressive loss of neurologic function. Dysarthria is a specific early indication of hypothermia and is often one of the first recognizable signs of the loss of deep body temperature. Judgement of the degree of frostbite has historically been a retrospective grading system involving four categories. It is more useful and realistic, however, to determine two major categories: superficial and deep. Because frostbite is a continuum of events, the differentiation between first, second, third, and fourth degree if often clouded and may take some days or weeks to become completely obvious. In first degree, erythema and edema along with transient tingling or burning are early manifestations. Skin becomes mottled blue or grey and red, hot and dry. Swelling begins within two or three hours and persists for ten days or more

depending upon the seriousness of the injury. Demarcation of superficial epithelium begins in five to ten days and may continue for as long as a month, but no deep tissue is lost. Parasthenia and deep aching and necrosis of pressure points of the foot are a common sequelae. Increased sensitivity to cold and hyperhydrosis may appear, especially in repeated first degree injury patients. It should be noted that it is difficult to differentiate first degree frostbite from abrasion produced by the insulated vapor barrier boot. Medical personnel must be cognizant of the difference as they both occur in the same clinical setting. Second degree cold injury starts as with first, but progresses to blister formation, anesthesia and deep color change. Edema may form, and disappear within days, but vessicles appear within 12 to 24 hours. They generally appear on the dorsum of the extremities and when these vessicles dry they form an eschar. Blisters are a good clinical sign as long as they are filled with clear fluid. If the fluid is hemorrhagic, it is not a good sign. As these vessicles dry they either sluff cleanly with pink granulation tissue beneath or they form black eschars. Throbbing and aching pain occurs three to ten days after this injury. Hyperhydrosis is apparent between the second or third weeks. Early rupture of the blisters with subsequent infection often occurs in second degree cold injury. This infection signficantly increases the severity of the frostbite injury. Third degree injury involves full skin thickness and extends into subcutaneous tissue. Vessicles are smaller and may be hemorrhagic. Overall edmea of the extremity may occur, although it usually disappears in five or six days. Subfacial pressure increases and compartment syndromes are common in third

and fourth degree cold injuries. If pressure rises significantly with loss of distal blood flow, faciotomy along with vasodilators are indicated for therapy. The skin forms a black, hard, dry eschar, usually thicker and more intense than the second degree injury. When it finally demarcates, sloughing occurs with some ulceration if there is no complicating infection. Average healing time is 68 days. Patients often complain of burning, aching, throbbing or shooting pains beginning on the fifth day and usually lasting through 4 or 5 weeks. Hyperhydrosis and cyanosis appear late and extreme cold sensitivity is a common post injury sequelae. In fourth degree injury, there is destruction of the entire thickness of the part including the bone resulting in extensive loss of tissue. After rewarming, the tissue is cyanotic, insensitive, with minimal blister formation and if present, they are hemorrhagic. Severe pain on rewarming along with deep cyanotic appearance regularly occurs. In rapidly frozen extremities or the freeze-thaw, refreeze injury, dry gangrene progresses quickly with mummification. With slower freeze, however, there is some early swelling and edema with deep pain, but it takes much longer for demarcation to occur. This line of demarcation becomes obvious at 20 to 36 days and extends into the bone in 60 or more days.

Management of Frostbite

Some degree of thaw almost always has occurred prior to medical attention. Superficial injuries are best described as exhibiting mild pain, tingling, numbness and some swelling. More severe injury appear cold, waxy, inflexible and the skin will not move freely over the joints. Patient will describe a painless injury early or a throbbing, searing pain upon

rewarming followed by significant edema, blister formation and eventually necrosis. It is sufficient medically to differentiate superficial from deep injury. Early prognostication is extremely difficult even among physicians with extensive experience in frostbite management. Prehospital care instructions should include keeping the frozen part away from extreme heat and careful handling of the extremities. The danger of freeze-thaw-refreeze in military setting is high and yields devistating results. Superficial frostbite can best be managed by rewarming with the hand or warm moist towels. If it is deemed severe, however, rapid rewarming in water, preferably whirlpool, between 37.8 and 43.3 degrees C is advisable. Higher temperatures may produce more serious injury. A pink flush will occur quickly. Patient will complain of extreme pain during this process. Blisters may form during the first 12 to 24 hours. Patient should be encouraged to conduct full range of motion exercise in the rewarming bath and all subsequent whirlpooling to prevent flection contractures. The deep red flush of initial rewarming may be followed by deep purple cyanosis. If proximal arterial pulses are lost, with indication of elevated compartment pressures, faciotomy may be indicated. If it is deemed to be a deep frostbite injury, Reserpine 0.5 mg. per cc injected interarterially, proximal to the injury has been shown to be extremely effective in producing peripheral vasodilation. Each limb must be injected separately and no systemic blood pressure changes occur if injected interarterially. Three indications for using reserpine are 1) a perceived severe injury 2) the pulsitile pain late in treatment and 3) persistent infection. Intravenous fluids should be started. Low molecular weight dextran has been

shown to be mildly effective in maintining blood flow in the frostbitten extremities. Persistent infections should be cultured, sensitivities taken and appropriate antibiotic therapy initiated. The limbs should be elevated, the whirlpooling should be done twice daily, and Reserpine injected as indicated by peripheral vasoconstriction. The limbs should be wrapped in sterile towels and elevated throughout the treatment process. Blisters should remain intact after the rewarming unless they become infected. Provodone iodine or hexachlorophine should be added to the whirlpool bath. Amputations earlier than six weeks should not be done. A 1 or 2 millimeter line of liquifaction occurs between viable and mummifying tissue and auto amputation is advisable. Early intervention often leads to loss of viable tissue, infection and retraction problems. Post injuries sequally including parasthenia, cold sensitivity, hyperhydrosis, causalgia, and bone and joint changes are not uncommon. Treatment is supportive and symptomatic.

Hypothermia victims present with different degrees of physiologic depression depending on their core temperature and the duration of their exposure. Cold suppresses metabolic function and oxygen demand which enhances long-term survival even of severely cold patients. Recognition of this survival potential is critical to the successful resusciation and everyone involved in medical treatment and evacuation must be cognizant of the phrase "No one is cold and dead, only warm and dead. Failure to respond to rewarming is the only criteria for death in hypothermia". Two major defenses against the cold are peripheral vasoconstriction and

shivering. Peripheral vasoconstriction reduces the skin blood flow decreasing both radiant and convective heat loss to the environment conserving core heat. Shivering is involuntary muscle activity that increases heat production. Cold diuresis which decreases circulating volume is the end result of peripheral constriction. Shivering produces significant metabolite production including lactic acid. The longer individuals are in the cold the more metabolically abnormal they appear on presentation. Dry land hypothermics who shiver violently and diurese for long periods of time and have drawn on their defenses against the cold, often show the most severe metabolic abnormalities whereas water immersion hypothermics who cool rapidly do not shiver quite as long and often show a quite normal electrolyte and pH picture. As cells drop below 30°C in an acid medium the sodium pump fails and potassium leaks out of cells and becomes available to move in the general circulation. As core temperature drops in the presence of acidosis and hyperkalemia, severe cardiac arrythmias occur. Battlefield wounds and hemorrhage in a cold environment lead to rapid hypothermia, which may be partially protective for irreversible shock in acute hemorrhage situations.

Field appearance of a hypothermic often reflects lowering of cerebral metabolic activity. They show a stumbling gait, incoordination, slurred speech, and a retreating inward psychologically. Their senses are dull, they are apathetic, they become drowsy, they appear more exhausted than the activity would warrant and this progress to unconsciousness. Disorientation, confusion, irrational judgment and poor decision making

ability pose a significant threat in a leadership setting because leaders are often under the same physical and cold stress as their troops. They may, in fact, be unable to recognize the signs and symptoms of hypothermia in people around them because they are suffering the same symptoms themselves. This can spell disaster for large numbers of troops.

Field Management of Hypothermics

The hypothermic is seriously metabolically depressed and in a state of suspended animation or a "metabolic icebox". For conscious victims, individuals must be stripped of their wet clothing, insulated, given warm sweet drinks and encouraged to do large muscle activity which will warm them up. Applying heat externally is an extremely difficult physiological problem. Conscious individuals will shiver, and start rewarming and if other muscle activity is added, they will warm up quickly. Replacement of fluids is essential to improve peripheral circulation and skin blood flow and improve cardiac output. Comatose individuals must be handled carefully as rough handling can produce ventricular fibrillation or standstill. They should have a patent airway, be carefully stripped of their wet clothes, insulated, and transported as rapidly as possible to definitive medical care. Positive pressure ventilation is advised but chest compression is not. Such compressions may produce ventricular fibrillation. The likelihood that compression of a cold stiff dilated heart of highly viscous cold blood, and a rigid chest wall producing much cardiac output is highly questionable.

Field rewarming procedures of the comatose individual are time consuming, compromise the rescue and are probably ineffective. A heated humidified oxygen rewarming device, if available, may be effective but it is certainly not a major heat input for the comatose hypothermic victim. Management through the evacuation chain involves improving cardiac output, decreasing blood viscosity, adding heat to the core, improving acid/base balance, and improving the hyperkalemia. Treatment of these parameters depends on the level of sophistication at each teatment site. Hospital management should include active core rewarming utilizing peritoneal dialysis, AV shunt or peripheral rewarming involving torso water immersion. Rewarming blankets are slow but may be the only rewarming device available. Volume replacement is essential to decrease viscosity and increase cardiac output. Low central venus pressures are advisable early and should be increased slowly as there is an indication of endothelial cell function and the ability to hold fluid in the vascular space. Lactate free and potassium free fluids are advisable as lactate conversion to pyruvate below 32°C by the liver is not occurring and severe hyperkalemia from temperature degraded cellular sodium pump increases available potassium. Fluid replacement, and glucose and insulin infusions will improve the severe hyperkalemia seen in many hypothermics. Sodium bicarbonate is indicated early for initial improvement in pH. However, overzealous correction is ill advised. Patient should be kept mildly acidotic throughout the treatment process. An improved respiration during resusitation will improve pH significantly . Antiarrhythmic drugs are contraindicated. Excessive manipulation early may lead to ventricular standstill which is

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